recorded, and, indeed, a mortality of 90 per cent. was not at all encouraging. But the sixteen cases subjected to operation during the second lustrum showed a death-rate of 87.5 per cent., while during the period from 1894 to 1898, inclusive, the mortality of the operation had been reduced to 72 per cent. This is probably still the death-rate, although of cases recorded since 1898 less than 70 per cent. have terminated fatally; but no statistician, however enthusiastic, can afford to ignore those cases, mostly fatal, which are never reported at all, and must, therefore, allow a fairly wide margin in drawing conclusions from the figures available.

We have carefully examined in all the records of 362 patients, who, during the course of typhoid fever, submitted to laparotomy for intestinal perforation, or for peritonitis without actual perforation. From our tables we have excluded several cases where an operation was not performed until some time after recovery from typhoid fever, and also a very few cases accepted by previous writers on the subject, in which study of the original records has convinced us that the evidence did not warrant their inclusion. We have freely consulted the works of Keen, of Finney, of Hagopoff, of Miclescu, and of others; also the theses of Mauger, of Junqua, and of others; as well as numerous articles of less extent, among which mention may be made of those by Shattuck, Warren and Cobb, Bontecou, Munro, Monod and Vanverts, Briggs, Cushing, Abbe, Shepherd, Hays, and Hartmann; to all of which we desire to express our indebtedness. We have consulted the original reference in every case in which it was accessible to us, and in other instances have taken pains to indicate the medium of our knowledge.

Perforation of the intestines is said to occur in from 1 to 11 per cent. of cases of typhoid fever. Leibermeister, in 2000 patients, found perforation in twenty-six; Murchison, in 1580 patients, observed it forty-eight times; Griesinger, in 118 noted fourteen perforations; while Flint, in seventy-three cases, found but two (these figures are from Hutchinson, in

Pepper's *System of Medicine by American Authors,* Philadelphia, 1885, Vol. i.). Curschmann, in Nothnagel's *Encyclopædia,* Philadelphia, 1901, found that of 829 patients 2.7 per cent. perforated. Armstrong, in the Montreal General Hospital (ANNALS OF SURGERY, November, 1902), observed among 932 patients an incidence of 3.66 per cent. At the Episcopal Hospital in this city we have examined the records since January 1, 1898, and find that, among 1556 patients treated for typhoid fever in that time, at least thirty-four perforations occurred; and at the Pennsylvania Hospital, among 1793 cases, forty-five perforations are recorded.

These figures may be compendiously seen in the annexed table:

Authority.	Cases.	Perforations.	Percentage.
Leibermeister	2000	26	1.3
Murchison	1580	48	3.03
Griesinger	118	14	11.01
Flint	73	2	2.7
Curschmann	820	22	2.7
Montreal General Hospital	932	34	3.66
Episcopal Hospital	1556	34	2.18
Pennsylvania Hospital	1793	45	2.5
Total	8881	225	2.54

CAUSES.

The causes of perforation are numerous. As those predisposing to this lesion may be discussed race, sex, age, season, geographical location, stage of the disease, severity of the attack, intestinal parasites, etc. It may in general terms be said that the white *race* is more disposed to perforation than is the negro race, but sufficient statistics on this point have not been collected to enable us to speak with certainty. The male *sex* is more liable to suffer perforation than the female in the ratio of about four to one. Of 279 cases in which the *age* is known, over 12 per cent. occurred in patients under fifteen years of age, over 54 per cent. between fifteen and thirty years, and only 33.6 per cent. over thirty years of age. *Season* and *geographical location* have little influence in predisposing to per-

foration, although, of course, more perforations are apt to occur in the summer and autumn months in this latitude, because there are more cases of typhoid fever at these seasons. It would seem, also, that in warm and semitropical climates this complication is somewhat more unusual than in temperate and cold climates; at least, the majority of reported cases occurred in the United States, Great Britain, France, Germany, and the northern parts of Russia, although cases have occurred in Italy, in Constantinople, and other southern climates. In Mexico it is said to be very unusual.

Intestinal parasites may act as a predisposing cause, and even rarely as an exciting cause. Most of the earlier cases of perforation of the stomach reported were due to intestinal worms, and it seems not improbable that many cases of intestinal perforation reported during the last part of the eighteenth and early part of the nineteenth century as caused by worms were, in reality, in patients suffering from typhoid fever, the physician's attention at the autopsy being drawn away from the mucous surface of the intestines by both the parasites and the extensive peritonitis. Out of 932 cases of typhoid perforation reported as above stated by Armstrong, two had intestinal worms. We have not ourselves observed this complication.

Stage of Disease.—Out of 286 cases in which is known the stage of disease at which perforation occurred, only six, or about 2 per cent., took place in the first week. The second and third weeks witnessed 162 perforations, or 56.6 per cent. of the whole number; while after the end of the third week only 41.2 per cent. occurred.

Severity of Disease.—It has been stated that perforation is more apt to occur in the mild than in the aggravated form of the disease. Dr. George B. Wood [2] expressed himself of this opinion; Curschmann says perforation is more frequent in ambulatory and mild cases, also among the lower classes, for the same reason, namely, lack of proper care; [3] and Osler says, "There is certainly no relation between this accident

and the severity of the disease."[4] But, on the other hand, Hutchinson[5] claims that in a large proportion of cases it occurs in grave cases, and Osler himself, in a later utterance,[6] reverses his opinion by stating that perforation usually occurs in the more severe cases. This is a point which it is difficult to determine; because many ambulatory cases would never be seen in hospitals at all, unless perforation had occurred; and because, when perforation does occur in very severe cases, it is often extremely difficult to diagnose on account of the apathy or hebetude of the patient; and it thus not infrequently happens that a perforation is unexpectedly found at autopsy, when it had been thought that the patient had died merely from the toxæmia incident to a prolonged course of the disease. Moreover, we recollect in after years chiefly those cases where the perforation comes contrary to all expectations; while those patients in whom its advent is hourly anticipated do not create so lasting an impression on our minds. We are, therefore, of the opinion that, although perforation certainly does occur most unexpectedly in some patients, and although in patients who have already reached the stage of convalescence, and in those suffering from an ambulatory form of the disease, its occurrence has frequently been observed, yet that in the majority of instances it is a lesion of the severer forms of the disease, and that it is in these cases that preventive measures, such as they are, should be employed. In connection with the severity of the disease, the existence of *tympany* as a predisposing cause of perforation may be considered. Where ulceration of the bowels is extensive and tympany coexists, there is every reason to believe that perforation is more apt to be produced by mechanical causes, such as a sudden turn in the bed, etc., than in cases where the intestines are comparatively flaccid. The condition of the intestinal walls themselves is, of course, of importance as a predisposing cause: attention has recently been called to the action of the thrombi so frequently observed beneath the serous coat of the bowels in typhoid fever, and it has been asserted[7] that they act as a factor favoring the occur-

rence of peritonitis without perforation, and, it seems reasonable to infer, also in predisposing to perforation.

EXCITING CAUSES.—The exciting causes are chiefly those which act mechanically. Anything, in fact, which sets up unusual peristaltic action is liable to cause a solution of continuity in the already damaged intestines; among these, indiscretions in diet probably hold first place; then purges injudiciously administered, a large or too forceful an enema, a sudden motion in the bed, straining on the bed-pan, the almost involuntary contraction of the abdominal muscles produced by the physical shock of the cold bath, each one of these and others have been held responsible for the immediate onset of symptoms of perforation.

PATHOLOGY.

The lesions of the intestine in typhoid fever consist, as is well known, mainly of changes in the lymphoid tissue found in greatest abundance in the lower part of the ileum. In the first week or ten days of the disease the intestinal lymph nodes are swollen, Peyer's patches being, as a rule, affected earlier than the solitary glands. This swelling consists, as has recently been shown by Mallory,[8] of Boston, chiefly in a proliferation of the endothelial cells of the lymph and blood-vessels; these endothelial cells show a marked tendency to gorge themselves with lymph cells (lymphocytes), thus constituting the pathognomonic "typhoid cells." It is to be observed that a large number of polymorphonuclear leucocytes does not collect until either a mixed infection has arisen or until the typhoid germs have taken on pyogenetic properties, so that in the early stages of the disease there is not usually an appreciable amount of "round-celled infiltration." If this medullary infiltration, as it is called, above described, does not resolve, the affected area is thrown off into the lumen of the bowel by a process of coagulation necrosis. This process is aided partly by the mechanical ischæmia produced by the choking of the vascular channels, and partly by the direct action of the typhoid toxines.

These toxines, spoken of rather vaguely in the past, have taken on a new significance from recent studies[9] which apparently show that they are the agents producing the agglutination thrombosis of the erythrocytes in typhoid fever. This thrombosis, as well as the ordinary form of thrombosis (produced when the engorged endothelial cells degenerate and induce a precipitation of fibrin,—a coagulation necrosis), predisposes to the sloughing, which is either molecular or massive. The surface left by the slough forms the ordinary typhoid ulcer, the floor of which, in the large majority of cases, is formed by the muscularis mucosæ.

Now, if this medullary infiltration has involved the whole thickness of the intestinal wall, when the slough is cast, naturally, a perforation of the bowel results. Accordingly, we find that perforations occurring at this stage of the disease are rarely of the pin-point size so frequently observed at a later date. The reports we have examined are not, most of them, circumstantial enough to enable us to derive any authoritative statistics with regard to the relation of the size of the lesion to the stage of the disease at which perforation occurred; but there can be little doubt that, as a rule, the large perforations occur early in the course of the fever.

If, on the other hand, the slough cast off is slight in depth, the resulting ulcer usually heals. The sloughs are usually separated about the end of the second or the beginning of the third week.

When the ulcer fails to heal and gradually deepens, as it approaches the peritoneal coat a slight degree of plastic peritonitis may be set up; the serous coat becomes congested, slight effusion ensues, a patch of lymph is formed on the serous surface, and, in favorable cases, the bowel becomes attached to a neighboring coil or to some part of the parietal peritoneum. This is Nature's method of healing a perforation. A specimen illustrating this process is in the Museum of the Pennsylvania Hospital. A tag of omentum has, in some cases, been found at operation attached feebly to the perforated area.

If the process be not arrested at this point, an abscess will form; and cases have been reported in which such an abscess was not opened for two or three months subsequently, the patients ultimately recovering.[10] If, however, no adhesions form, then perforation into the free peritoneal cavity takes place, and general peritonitis results. Even in cases of perforation arising after this manner, the size of the bowel opening may be great, or several small perforations may form in the floor of the same ulcer. At times it almost seems as if the stage of medullary infiltration persisted throughout the disease, since in cases of perforation from ulceration the surrounding parts of the perforated Peyer's patch sometimes are so thickened and friable that it becomes impossible to make sutures hold; whereas at other times the bowel has become so thinned by extensive ulcers that it appears like paper. At times, too, it is impossible to tell whether the perforation has arisen from sloughing or from progressive ulceration.

Much has been written about a preperforative stage; and there can be no question but that there is always a time previous to perforation, but we very much doubt whether it can be recognized as a pathological any more than as a clinical entity; because so often there may be a certain degree (and at times a very marked one, too) of peritonitis without any macroscopic evidence of perforation.

As to the form of the perforation, the larger circular lesions are, as has been stated above, generally due to sloughing; the smaller, or cribriform, to ulceration; and the oblong, slit-like perforations have been thought to be due to traumatism.

The perforation is usually situated on the part of the bowel diametrically opposite to the mesenteric attachment, where the lymph glands of the intestine are found and where the blood supply is poorest. Occasionally, however, an ulcer will perforate between the layers of the mesentery, and a retroperitoneal abscess will be formed. Such a lesion may be mistaken for a suppurating mesenteric gland.

The perforation is fortunately single, as a rule; in 271

eases in which this point is mentioned, a single perforation occurred in 236, while in the remaining thirty-five it was multiple. In seventy-two eases the perforation was less than one-eighth of an inch in diameter,—practically pin-head in size; in seventy-three cases it was under one-half an inch; and in only twenty-three eases was it over a half-inch in diameter,—there being only 168 eases in which the size of the perforation was mentioned.

The site is mentioned in 190 eases, and in 140, or over 73 per cent., of these the lesion was found within twelve inches of the cæcum; and in only four eases, or 2.1 per cent., was it more than three feet distant from the ileocæcal valve. In seven eases only was the colon perforated, five times the ascending colon, once each the transverse colon and the sigmoid flexure. Meckel's divertieulum was perforated three times, and the appendix eight times. It is to be noted that we have carefully excluded from this series every case of perforated appendix in which the lesion has not undoubtedly been proved to be of typhoid origin, even rejecting, as of doubtful value, some such cases previously accepted by other authors. There were twelve patients in whom a subsequent perforation occurred. These all died. In three eases, while one or more perforations were found at operation, yet others in the same patients were not detected,—these all likewise perished.

At times the perforation is only to be seen after a patch of lymph has been removed from the bowel, the perforation then appearing as a dark spot in the centre of an intensely congested area, at times nearly sphacelated and falling away beneath the fingers.

There was not a sufficient number of eases reported in detail to enable us to determine in what proportion or after what lapse of time adhesions would be found; but, generally speaking, it may be said that adhesions are the exception, that they are usually indicative of a mixed infection, and that, except in eases of several days' standing, they are of unfavorable prognostic import. With regard to the bacteriology, it has been found that in eases where the typhoid bacillus alone

is the infecting cause, the usual lesions are a low-grade peritonitis, frequently lemon-colored exudation, few adhesions, and not much lymph. Where the streptococci or the staphylococci abound the lymph is more abundant, and adhesions are the rule if the peritonitis has lasted more than a few hours. The prognosis is much more grave in the streptococcic infection than in the typhoid. An interesting case in this connection was observed at the Johns Hopkins Hospital. At the first operation on a patient suffering from typhoid perforation bacteriological examination of the peritoneum showed no organisms, while the intestinal contents contained streptococci in abundance; this patient was later subjected to a second laparotomy, at which time the peritoneum was found to be infected by the streptococci, and death soon followed from the peritonitis.[11] Evidently in this case the time which elapsed between the initial symptoms of perforation and the operation —five and a half hours—was not sufficient to infect the peritoneum extensively with intestinal contents; whereas forty-three hours later, when the second operation was performed, general purulent peritonitis was well advanced.

SYMPTOMS.

In what may be called a typical case, the symptoms are well marked and easily distinguished; but, unfortunately, such a case is the exception. It is well known, moreover, both that a patient who has presented all the clinical evidences of a perforation may submit to laparotomy, and no perforation nor even a trace of peritonitis be found; and that, on the other hand, patients may die without any abdominal symptoms of importance, and at autopsy a perforation may be found, to every one's complete surprise.

But there are undoubtedly certain symptoms which should at once throw the attending physician on his guard, and which should make him disposed to consult one with surgical acumen, even before he is sure a perforation exists. A patient in the second or third week of the disease, who at various times has had slight or more severe stabbing pains in the abdomen;

whose abdomen is much distended and tympanitic; who, perhaps, is somewhat apathetic; who is apt to have retention of urine, and has perhaps lost control of his fæcal evacuations,—such a patient should be watched hourly. Any one of these slight stabbing pains may be significant of perforation, and, although pain alone is by no means pathognomonic, it is by all means the most valuable subjective symptom.

Pain.—The pain is, as described, usually a stabbing sensation, most frequently in the right lower quadrant of the abdomen; though for it to be felt in the epigastric or the umbilical regions is also customary, and a not unusual situation is in the bladder or, in the male, at the end of the penis.[12] Cases have occurred in which, on such complaint from a ward patient, the orderly has been allowed to use the catheter, drawing perhaps a couple of ounces of urine; and the patient, not again complaining, has been neglected, until in a few hours a fatal peritonitis has developed. But the pain may not cease so readily, and the patients at times will scream out and roll around the bed in an agony, doubling themselves up with abdominal pain, which may persist for two or three hours with undiminished intensity. Again, as has been mentioned, in a very apathetic patient no pain at all may be felt, or, at least, none be complained of. Or, on the contrary, there may be only a gradually increasing discomfort in the abdomen, with no sudden sharp onset of pain. This is more usual where the abdomen is already tympanitic and where peristalsis has been for some time very slight.

Vomiting occurs so frequently in severe cases of typhoid fever with no apparent exciting cause that it cannot alone be considered at all indicative of perforation; but where it occurs for the first time, and especially where it either precedes or follows pain, it should not be passed lightly by. If it is at all violent it will very likely cause a perforation, and in very many instances follows it.

Temperature, etc.—At the same time as the pain, or soon after, sweating may occur; it has even been known to precede the pain, which has always been supposed to be indicative of

the moment of perforation. With the sweat, or when sweating is absent, occurs a fall of temperature: a drop of four to six degrees is not infrequent. It has been claimed that this fall of temperature—this *hypothermie*—is not at all constant; and the dictum of Dieulafoy, "No perforation without fall of temperature," has been called in question by some authors, notably by his countryman, Lereboullet, who maintains, on the contrary, that a rise of temperature invariably follows perforation. Hagopoff tries to reconcile these statements by claiming, and, we think, not without reason on his side, that where no "hypothermie" has been observed it is simply because the temperature was not taken sufficiently soon after the perforation occurred, but at a time later, when the temperature of the patient had risen as a result of the subsequent peritonitis; this explanation really upholding the views of Dieulafoy. Lereboullet's contention certainly cannot be true in all cases, since sometimes the patient is so collapsed as a result of the perforation that the ensuing peritonitis, even if he live long enough for it to commence, is not sufficient to raise his temperature to the normal, but he dies in collapse.

Very confusing in this connection are those cases in which several falls of temperature have occurred within a day or so of the suspected perforation. Fall of temperature is, of course, a frequent accompaniment of intestinal hæmorrhage during typhoid fever; and, either as a result of that, or even with no ascertainable cause, the patient may for several days, perhaps merely from general loss of vascular tone, have suffered at irregular intervals from sweats and sudden falls of temperature. So that neither of these symptoms, any more than abdominal pain, is to be regarded as pathognomonic of perforation.

Frequently a change in the solubility of the *bowels* follows perforation. Were they loose before, they become confined; or were they costive previously, this condition is replaced by diarrhœa.

Delirium may have pre-existed, or may develop only with the peritonitis.

Leaving now the subjective symptoms, we come to the physical signs, on which more reliance is to be placed; and of these by all means the most valuable is rigidity of the abdominal walls.

Rigidity.—By this we mean, not that rigidity which the physician may any day produce by thrusting his fingers into the patient's belly so as to feel the backbone, but the well-known rigidity ensuing on peritoneal inflammation, and which is involuntary, reflex, not produced by palpation. This muscular rigidity is most frequently observed in the right rectus muscle; also in the right oblique muscles and in the left rectus; in time spreading with the peritoneal inflammation all over the abdomen. A surgeon's rigidity and a physician's rigidity are two vastly different things, and the distinction is well worth remembering. The rigidity of pleural irritation is sometimes mistaken for peritoneal involvement, or *vice versa*. In one of our own cases operation was delayed because of evidences of a pulmonary lesion, whereas perforation was really present, as was sadly manifested by the peritonitis which developed a few hours later, and the operation performed then was too late.

Pulse and Respiration.—Along with these cardinal symptoms—pain and rigidity—there is, in the vast majority of cases, an increase in the pulse-rate. In most cases of typhoid fever the pulse is slow in relation to the height of the fever, rarely averaging more than 100 beats per minute; but after perforation the rate rises to 120 or 140 in a very short time, often within fifteen or twenty minutes; and the respirations may be accelerated as well. Not until peritonitis is well advanced does purely costal breathing replace the costo-abdominal, which is the normal; and patients who are at all conscious alter the character of their respirations so readily during examination that too much reliance should not be placed on this sign.

The facial expression is important. Almost from the very moment of perforation there is a distinct change of expression, difficult to describe, yet easily recognized when once seen. It is not the peritonitic facies, which closely approaches the Hip-

pocratic in type, but is distinctly characteristic of the shock of perforation. It is what the French might call an "abattement" of the countenance, consisting in a general weakening of the expression.

Tenderness, while often present throughout the disease, is probably, without exception, increased by a perforation. In appendicitis it may persist when, the appendix having become gangrenous, rigidity has disappeared; but we are of the opinion that in intestinal perforation rigidity is the more valuable sign of the two; for, as has been mentioned above, in the apathetic state to which typhoid patients often attain, tenderness sometimes cannot be elicited. When present, it is usually found in the right lower quadrant, near the customary site of the lesion.

Dulness to percussion is a very uncertain sign. It may exist from a fæcal accumulation, and be mistaken for the matting together of intestines from peritoneal inflammation. Even movable dulness in the flanks may be caused by fluid fæces within the intestinal tract, when there is no suspicion of perforation. But if well-marked movable dulness in the flanks develops in the course of an hour, as we have seen it do, it presents very excellent evidence of a solution of continuity in the intestinal walls, as it is scarcely conceivable either that fluid fæces within the bowels should at one time produce no such signs, yet within an hour later do so; or that an amount of exudation sufficient to give movable dulness could be formed by the peritoneum in so short a space of time. But, on the other hand, the absence of this free peritoneal fluid should, on no account, be taken as showing that neither perforation nor peritonitis existed.

The same remarks apply to obliteration of the liver-dulness as to movable dulness in the flanks; it may occur merely from tympany, without perforation, and even with perforation and with air free in the peritoneal cavity, the dulness over the liver may persist. However, they are both interesting signs, and it is well to note their presence or absence.

Œdema of the abdominal walls is occasionally observed

in this as in other intra-abdominal lesions, but has no specific significance.

Blood.—The condition of the blood is one of the most vexed questions that arises in this connection. It has been much more extensively studied by American than by English or Continental observers. Osler says that a steadily rising leucocyte count is indicative of perforation; for such an observation the white blood-cells should be counted every half-hour or hour during the period of uncertainty. Few hospitals, and no one in private practice, can afford to have this done, even the routine work in most institutions consuming the whole time of the clinical pathologist. In most cases where, in the course of twelve or eighteen hours, three or four examinations at the outside are made, the majority of advocates of the benefits conferred by blood examination claim that such erratic observations are of no use. The average surgeon is averse to having his diagnosis made for him in the laboratory, and will not hesitate to follow his own judgment, in the face of overwhelming contraindications from the microscopist's standpoint.

A wave of leucocytosis has been described, reaching its maximum soon after perforation, and then again subsiding. Differential counts have been claimed to be more reliable than a mere enumeration of the white cells as a whole, inasmuch as in commencing peritonitis there is a disproportionate increase in the polynuclear neutrophiles.

In drawing conclusions from any leucocyte count in typhoid fever, the normal leucopenia of this disease should be borne in mind.

This whole question is as yet undecided, the accurate observations being still too few to draw any very definite conclusions.

DIFFERENTIAL DIAGNOSIS.

A differential diagnosis has most often to be made from hæmorrhage. While the collapse is very likely the same in either case, yet hæmorrhage is, as a rule, not attended by pain, and there is no rigidity present; in hæmorrhage the blood is

generally passed by the bowel in the course of an hour or so at the outside; and in most cases where confusion is liable to arise, the occurrence of previous hæmorrhages in the same patient would incline one to pause before undertaking an exploratory laparotomy. Osler and others have reported series of cases in which attention is called to the frequent association of hæmorrhage with perforation; and in any case where reaction from the collapse, due to an obscure abdominal lesion, does not occur promptly, we are in favor of operative treatment, since in one of our own cases the bleeding point has been found by laparotomy and ligated.

The diagnosis from peritonitis without perforation is immaterial, as operation is indicated in both conditions.

From appendicitis the diagnosis can best be made by recollecting that in appendicitis there is rarely such collapse as occurs from perforation of the small or large intestine, even if the appendix he perforated. Vomiting is more apt to have occurred with the initial pain in appendicitis than in perforation. Moreover, vomiting may cause perforation, while it could scarcely alone produce appendicitis. Symptoms resembling those of appendicitis are not unusual at the beginning of an attack of typhoid fever, and in a number of cases the appendix has been removed under such circumstances with happy results. The differential diagnosis here, too, is not especially important, as the removal of the appendix for disease is indicated in typhoid fever as well as at any other time.

From peritonitis due to pelvic or ovarian disease the diagnosis can usually be satisfactorily made by attention to the history of the case, together with a vaginal examination.

We have seen intussusception occurring during an attack of ambulatory typhoid fever, but, as the peritonitis was already well advanced, no question of differential diagnosis arose.

Affections of the gall-bladder occurring during typhoid fever from infection by the typhoid bacillus are not uncommon; they present pain in the right hypochondriac region; sometimes jaundice; vomiting is more persistent; no fall of temperature occurs; there is little shock; and the physical

signs—tenderness, rigidity, dulness, palpable mass—aid in the localization of the disease.

Gastric ulcers may perforate during typhoid fever: the localizing signs here, too, are our chief guide.

Finally, suppurating mesenteric glands, or splenic infarcts, may give rise to peritonitis in this disease; but here the symptoms are those of peritonitis pure and simple; there are none of those characteristic of intestinal perforation.

PROGNOSIS.

It may be confidently said that of those patients who die after an operation, in very few, indeed, has the operation hastened death. Even in those cases which end unfavorably, it is usually observed that the patients have left the operating-table in better shape than that in which they were brought to it: so much is gained by intravenous saline injections, by vigorous hypodermatic stimulation, and frequently by the free douching of the abdominal cavity with hot saline solutions.

Only five of the patients operated on are known to have died before the conclusion of the operation. Of those that died, twenty-one lived two days, twenty-three lived three days, twenty-one lived five days, seven lived nearly a week, while eleven lived over a week, three over two weeks, and four over a month; certainly in these last eighteen cases the patients may be said to have recovered from the operation.

Of the whole number, 26 per cent. finally left the surgeon's hands well. A small proportion of these, to be sure, might look forward to an intestinal obstruction arising at any time of indiscretion in diet, if they had been the subjects of extensive peritoneal adhesions; some, furthermore, when last reported had still a fæcal fistula in the wound; and probably a considerably larger number had or would soon develop a ventral hernia; but taking them all in all, they had reason to be thankful to have escaped a typhoid perforation with their lives.

The extensive analytical tables attached show in compact form the mortality rate considered from very various points

of view. Briefly stated here, the most favorable cases have been those in girls from ten to fifteen years of age, the perforation occurring in the first week of the disease, when the constitution is still strong, or in convalescence, when the frame is already re-established; who have been operated on in the third hour after perforation, the single perforation being pinhead in size (under one-eighth of an inch in diameter), within a few inches of the cæcum, or in the appendix; and where neither fæcal extravasation had occurred nor adhesions were present. Such would be ideal cases, and the mortality should be less than 50 per cent.

TREATMENT.

The treatment of all patients suffering from intestinal perforation in typhoid fever should be by laparotomy and suture of the perforation; to trust to medical treatment alone is nothing short of folly. Some cases may be thought an exception to this rule: those patients who are manifestly moribund should be given the benefit of doubt, and if, under vigorous stimulation, any improvement is noted, or even if they cease to grow worse, the surgeon should not hesitate to interfere. The average time consumed in the operation by skilful operators is about fifteen or twenty minutes. Nor should it be forgotten that ether is a heart stimulant, and if the pulse improves under its administration operation is surely justified. We believe that well-authenticated cases of recovery from intestinal perforation without operation are excessively rare; indeed, since it has happened with every symptom of perforation present, and the abdominal cavity having been opened and explored, that no signs of perforation, much less of peritonitis, have been found, it may be considered proved that the examples of so-called recovery from perforation without operation are cases of this nature, and that while the symptoms of perforation and of peritonitis may have been present, yet such lesions did not exist. Even in cases, almost unknown as they are, where the patients have survived the immediate effects of the perforative peritonitis, yet, even many months subse-

quently, they have submitted to laparotomy for an abdominal abscess of apparently obscure origin, but evidently arising from the former peritonitis—the condition being, in fact, one of residual abscess. Hagopoff, seemingly a careful observer, accepted as genuine instances of recovery without operation only fourteen cases, rejecting those observed by Griesinger, which are recorded in the thesis of Morin. When it is recollected that, according to the calculations of Briggs,[13] more than 16,000 perforations of the intestine due to typhoid fever occur annually in the United States alone, the exceeding rarity of recovery without operation is appreciated by realization of the fact that for all time, and all over the world, only fourteen cases are known which have so terminated.

From the moment of diagnosis until the time of the operation little is to be done in the way of treatment except by stimulation, hypodermatically, or with intravenous saline injections. To avoid the delay sometimes occasioned to obtain consent of the family to an operation, it is well to arrange such questions, in every possible case threatening perforation, even before the diagnosis is made: the less time that elapses between diagnosis and operation the better for the patient.

Anæsthetic.—As to the danger of a general anæsthetic in such patients as these, we believe that while some operators may have the faculty of so hypnotizing their patients as to make local anæsthesia available, yet the average surgeon will succeed better by the use of ether judiciously administered. In other climates, and in higher altitudes, we have no doubt that with care chloroform can be safely employed, but certainly in the middle and northern parts of the United States nothing is so satisfactory as ether. We are, moreover, of the opinion that a general anæsthetic will, in the average patient, tend to diminish shock; although we freely admit that the abolition of all afferent impulses produced by the cocainization of sensory nerves will theoretically render impossible the physical conditions now believed to be necessary for the production of surgical shock. But mental perturbation is at least an equally disquieting element for the surgeon to deal with, and

this is in no respect diminished, but rather augmented by the consciousness on the patient's part of the details of the surgical procedures.

Most of the patients with perforation seen by us have been already in such a state of prostration that only a few whiffs of the anæsthetic have been required to completely anæsthetize them, as well as to render them unconscious and to relax their abdominal muscles, a state which is not attained by the use of a purely local anæsthetic.

In the majority of cases where the operation has been commenced with a local anæsthetic, it has been found expedient to resort to the use of ether or chloroform soon after opening the peritoneal cavity, and often fifteen or twenty minutes have been consumed in making the incision through the abdominal parietes alone, the patient meanwhile complaining bitterly. This state of affairs, we should add, has not always been the case, for some surgeons have had most gratifying results from local anæsthesia in operations of this kind.

When anæsthetization is begun (and this should not commonly be done until the patient is on the operating-table), the abdomen should be shaved, including the pubic hair, and cleansed. We prefer for this purpose turpentine, followed by green soap and water and then by alcohol; the whole area is then thoroughly rinsed off with corrosive sublimate (1 to 2000) and covered with a cloth wrung out of this solution until the surgeon, who has meantime been cleansing his hands, is ready to operate. The three processes—anæsthetization, cleansing of the abdomen, and washing of the surgeon's hands are commonly completed at about the same time, so that everything is coincidently ready for operation. It is convenient for the surgeon to have two assistants as well as three nurses at his service: one nurse to hand instruments, thread needles, etc.; a second to attend to the gauze sponges; and a third to bring hot salt solution, etc., as wanted. There should, likewise, be a fourth nurse at the call of the anæsthetizer to administer hypodermatic injections, tend the oxygen apparatus, and similar duties. Where facilities abound, a third assistant for

the surgeon and a fifth nurse can frequently be advantageously employed in giving an intravenous injection of hot normal salt solution; seldom more than three pints are necessary, often two is an abundant quantity. By recommending all this array of assistants as convenient, we do not by any means wish to be thought to discountenance impromptu surgery where necessary in these cases. There have been brilliant successes reported from operations done by lamplight on a kitchen table, in huts miles away from city hospitals; and such records are among the proudest in surgery; but wherever possible it is the operator's duty to organize success.

The cleansing of the abdomen, while thorough, should not be vigorous, both for fear of diffusing any extravasated fæcal matter and of producing further perforations.

From the time the knife is first taken in hand things should move with rapidity; this is only possible by system and regularity. We know of no other condition, except it be, perhaps, hæmorrhage, in which speed in operation is so imperative. In too many instances it is a race with death, and there are often anxious moments when it is questionable who will win; yet in only five of the reported cases is death known to have occurred before the operation was completed. A "death on the table" is, under all circumstances, a most painful occurrence in surgery, but it is doubly so if the surgeon has in any way to blame himself for delays that might have been obviated by a prudent mind.

The incision is best made to the right of the median line, over the cæcal region. Many of the earlier operators employed a median incision, in most instances hypogastric, but in not a few epigastric; their results, however, and especially so with the epigastric incision, do not invite imitation. In ninety-six cases where a median hypogastric incision was employed the mortality was over 78 per cent., while in 141 cases where the right lateral incision was used the mortality was less than 70 per cent. Of course, were an abscess to be manifestly pointing in a certain region it would be absurd to open the abdomen elsewhere. Where the median incision has been

employed, it has only too often happened that the surgeon has searched the presenting small intestine diligently without finding a perforation, until he has unexpectedly lighted on the duodenum, and has then been forced to retrace his steps downward to the cæcum, thus losing valuable time, and shocking his patient by prolonged manipulation of the bowels.

The length of the incision need not in abdomens of average thickness exceed four inches, as a rule; in uncomplicated cases three inches can be made to suffice; while in those patients with immensely fat abdominal walls the incision may, unfortunately, have to be extended nearly to the edge of the ribs upward and to the symphysis below. It is, on the other hand, by no means wise to endeavor too much through too small an opening; much harm may occur from blindly tugging at a coil of intestine which fails to come out through a wound, not because the latter is too small for such delivery to be physically possible, but because the bowel is retained by adhesions, the incautious rupture of which may cause much mischief.

The incision we prefer is that through the outer half of the right rectus muscle: it is straight, simple, direct, and economical of time; the deep epigastric artery is not apt to be wounded with ordinary care, using the handle rather than the blade of the scalpel to separate the muscular fibres; it is a route that gives direct access to our landmark, the cæcum, and to that part of the small gut most often affected; it affords quite sufficient room for exploring nearly the whole abdomen; it can be extended in either direction at need, and offers excellent drainage facilities. In those very rare cases where the perforation is in the sigmoid or in those parts of the small intestine not in reach of this wound, this incision should be temporarily closed, and another made where, from intra-abdominal exploration, it is evident that it would be nearest the seat of the trouble.

When the peritoneal cavity is opened the cæcum should be located; it is usually immediately beneath the wound or a little to the outer side. The vermiform appendix should be inspected and removed, if necessary. If extravasation is free

no packs are required. If, however, it appears that the peritonitis is fairly well localized the affected area should be walled off with large gauze pads. The ileum is next drawn into the wound and carefully examined; it is a great advantage to have two pairs of eyes examining it at the same time as it passes through the surgeon's hands, one watching the upper and one the lower surface. It is usually inadvisable to replace the gut at once on withdrawal, as this consumes valuable time, which is better spent searching for the perforation until found. If, however, more than two or three feet of the ileum have been examined fruitlessly, and there still appears to be reason to consider a perforation present, the eventrated parts should be re-examined, commencing at the highest point withdrawn, and ending at the cæcum, the coils being replaced as the return is made, but the highest coil being kept out of the belly until all the others have been replaced, as a starting-point for further search, which should proceed upward along the ileum with like manœuvres until there appears no further prospect of finding a perforation. This probability decreases as the area of greatest inflammation is left behind. The appearance the perforation will present can often be fairly accurately foretold by attention to the known pathology of the disease: if fæcal extravasation is free a medium or large-sized hole may be expected; if lymph abounds and clings to the bowel in patches, frequently beneath one of these patches will be found the pin-head lesion as a dark spot in the middle of an intensely congested Peyerian gland.

Throughout these manipulations the utmost care should, of course, be exercised to prevent chilling of the eventrated bowels. This is best secured by covering them with a hot towel, and constantly douching it and the bowels with hot saline solution; this aids, too, in washing away pus, etc. It should be seen, also, that the patient is not thus made to lie in a pool of rapidly cooling water; he should be so protected by blankets, mackintosh cloths, etc., as to maintain his bodily warmth, and, if necessary, he may even be surrounded by hot-water cans during the entire operation. If adhesions are

present, the operation is much more tedious and difficult; but fortunately they are rare.

Suture.—When the perforation is found all other coils of intestine should be replaced, and it should be sutured. But if the bowel is much distended with gas or fæcal matter, it is well to allow it to discharge itself through the perforation outside the abdomen. Indeed, puncture of the bowels for this purpose has been employed to facilitate eventration, but we have never found it necessary to do this, although, to aid their reposition, it is sometimes nearly imperative. Any such puncture is easily closed with a couple of Lembert sutures.

Excision of the ulcer we deem not only perfectly useless, but harmful as well, since it consumes unnecessary time and might open a blood-vessel.

Black silk we think the best material for the intestinal suture, using it threaded on a round straight needle. Such a needle is more easily controlled by the fingers, and, when time is such an important element, such devices as needle-holders may well be dispensed with. Usually we prefer to apply first a row of mattress sutures parallel to the long axis of the gut, crossing the longitudinal Peyer's patch at right angles. A second row of Lembert sutures, likewise applied longitudinally, but continuous, is also employed; and in the vast majority of cases these two tiers are quite sufficient to occlude the perforation satisfactorily. At times it may be more expedient to use a purse-string suture, the application of which takes less time, and which holds well if the perforation is small and the surrounding bowel in fairly good condition. We seldom rely on Lembert sutures alone, even in several superimposed rows. We avoid, where possible, a line of suture transverse to the long axis of the gut, believing that the tension on the stitches is thus greater and that the lumen of the bowel is usually more impaired. It is unusual to find the diseased area extending as far transversely as it does longitudinally, but where this is the case in such a degree as to make suture impossible, or from other cause, the grafting of a piece of omentum over the defect, enterectomy, with end-to-end anastomosis, or simply the estab-

lishment of a false anus, must be considered. The choice depends largely upon the general condition of the patient, the skill of the surgeon, the location of the perforation, and the state of the peritoneal cavity. To make a false anus consumes less time and is easier of operation to one unaccustomed to abdominal surgery. It may even be done without suture by the judicious use of gauze packs, leaving the perforation isolated and beneath the abdominal wound. Of four cases treated by the establishment of a fæcal fistula, every one died. This tremendous mortality, however, does not prove that the operation is necessarily fatal in cases of typhoid perforation, but merely that it has been employed only in exceedingly ill patients. On the other hand, of those sixteen patients who withstood the immediate shock of the operation, and in whom a fæcal fistula developed in the wound after operation, only two died,—the surprisingly low mortality of 12.5 per cent. This shows that direct drainage of the intestinal tract is actually beneficial. In this connection it is interesting to recall the treatment by the establishment of a false anus advised by some authorities in cases of colitis, and even in typhoid fever, as a means of drainage and irrigation of the large bowel.

But the establishment of a fæcal fistula is sure to prolong the healing of the wound, and, in a fair proportion of cases, will require a subsequent operation for its closure. In those exceptional cases where the lesions are high in the intestinal tract, it is, of course, not to be considered, on account of the well-recognized danger of starving the patient by short-circuting his ingested food. Moreover, it is difficult to drain the peritoneal cavity effectually through the same wound at which the bowels are emptying themselves, and there is the constant risk of reinfecting the peritoneum by leakage backward. So that, on the whole, where inversion of the perforation by suture or omental grafting is impossible, excision of the diseased portion of the gut, with circular enterorrhaphy, is by all means to be preferred. But this should not be lightly undertaken; for while cases have been recorded where the narrowed lumen of the bowel, due to inversion of the perfora-

tion, has been held responsible for death from intestinal obstruction, yet we believe that this complication is not to be apprehended, except where practically no lumen at all exists: the fæces in the ileum are usually fluid, and there is no reason to expect that a stricture thus formed may not dilate sufficiently in the natural course of events to cause no further trouble.

Circular enterorrhaphy may be performed satisfactorily here without mechanical appliance; those who prefer such devices may employ the O'Hara or the Laplace forceps, and the Murphy button may be used without fear of evil consequences. It has been thought that this last would be liable to cause a new perforation while passing out, or would pull loose from the friable intestinal walls by its own weight; but we do not think these accidents more liable to occur in typhoid fever than in other cases. Intestinal resection was employed five times in our series of cases, with only one recovery. The Russian surgeons have been its chief advocates, but it is apparently too severe an operation to be usually recommended.

Omental grafting is applicable as well to ulcers threatening perforation as to actual perforations. The omentum is to be adjusted over the defect and retained in position by points of the interrupted suture. In some cases the omentum has been found already covering in the perforation and preventing fæcal extravasation. Grafting the omentum in place is in every way preferable to either establishing a false anus or to enterectomy.

In selecting the special method of operation time should not be unnecessarily wasted; every surgeon knows that it is better to do the second-best thing for his patient than to let him die while a decision is being reached.

Every perforation having been found and treated as seems indicated, the *toilet of the abdominal cavity* should next claim the surgeon's attention. While he retains the sutured gut beneath the wound, he should, in our judgment, if there has been fæcal extravasation or if there is general peritonitis, resort to free douching of the abdominal cavity. And in doing this he should remember that the perforation is now practically out

of the question, and that his only hope in saving the patient lies in combating the peritonitis. To this end a large tube, one-half inch in diameter, with multiple perforations, should be introduced through the wound, a rubber tube and a funnel being attached to its outer end, and through this should be poured gallons of hot solution. We are of the opinion that the quantity has more to do with success than the quality of the fluid used, although our own practice is to use normal salt solution in ordinary cases; and in cases with much pus, streptococcic in origin, we use first the normal salt, then equal parts of normal salt and hydrogen peroxide, and, finally, normal salt again—several quarts of each solution being employed. The temperature of these solutions should be at least 110° F., and to insure its reaching the abdomen not below this temperature, it is well for the thermometer in the receptacle from which the supply is drawn to stand at not less than 115° F. The tube through which this douching is carried out should first be applied to the pelvis, in the rectovesical space, and in females both in front of and behind the broad ligaments; it should then be carried to the left flank and loin, up even to the spleen; the right side should be similarly treated, and the spaces between the intestinal coils should finally be visited. The douching should be continued until the return fluid is clear. Some surgeons have advocated first sitting the patient up to allow all matter to drain into the pelvis, and when that had been accomplished, reversing the body into the Trendelenburg position, and then mopping out the pelvic cavity with gauze. A patient treated by these heroic manipulations recovered.

When the surgeon is satisfied that the abdominal cavity is as clean as he can make it by this method, but not before, the tube may be withdrawn, but the main part of the fluid that has not already escaped, and which is clean, should be allowed to remain.

Several of the earlier operators used carbolic acid or boric acid solutions for this douching; and, as we said before, we do not think the composition is of any particular moment, the

cleansing being largely mechanical. Some of the later surgeons, notably those at the Johns Hopkins Hospital, have, under local anæsthesia, prolonged this douching for upward of an hour, but we do not think much is gained by such a course. It is, however, unquestionable that very many patients show very marked constitutional improvement from the abdominal douching; the heat combats the shock, and the fluid is constantly absorbed, thus taking the place of or aiding an intravenous saline injection.

Many authorities are unalterably opposed to douching the abdomen at all in these cases, asserting that the infectious particles are only more widely diffused, and relying entirely on dry sponging and rubbing off of lymph patches, etc. While we have no desire to deny that these methods have in some instances been followed by the happiest results, and while we have ourselves treated cases without douching of any kind, yet we remain firm in our belief that by douching in the manner above described lies the surgeon's greatest hope of success. Moreover, we recollect that long ago Sir James Paget contended that "wounds," and, we may add, *a fortiori*, the peritoneum, "should not be scrubbed, even with sponges."

In the appendix will be found an analysis of those cases where the description of the operation was sufficiently minute to enable us to abstract the treatment in regard to irrigation, wiping, drainage, etc. It is to be regretted that most reports are so vague in these particulars as to be not adapted for such purposes. Those cases marked "wiping and drainage," with a mortality of 61 per cent., were mostly cases of purely localized peritoneal lesions, as were also those where neither irrigation nor drainage was employed, with a mortality of only 41 per cent.; so the success here was only relative, and does not show that these methods are the best for all cases.

Drainage.—Drainage is necessary in nearly every case. We now prefer gauze drains to the tube we formerly employed. The amount and the extent of the drains to be inserted will, of course, depend on the extent of the peritonitis. In the simplest cases a wick of gauze alone may be placed to

the site of the sutures in the intestine, and the abdominal wound closed through nearly its entire length. If, however, there be any suspicion of pelvic involvement, a good-sized strip of gauze should be carried down to the pelvic floor. We commonly employ pads of sterile gauze, about six by eighteen inches in size, composed of four thicknesses, stitched together; or loose, crumpled gauze answers equally well, although not so handy. We see no advantage in iodoform or other medicated gauze in these cases, and never employ it. Some instances of poisoning from iodoform gauze indiscriminately used in abdominal surgery are within our cognizance, and the possibility of such an event alone is enough to deter one from its use. In cases of wide-spread peritonitis a second piece of gauze to the left flank, a third to the right, outside the descending and the ascending colon, respectively; a fourth above the ileum and to the inner side of the ascending colon, are advisable, and several more as may be required to hold the small intestines away from the pelvis and the site of operation. We have seldom had to use more than seven such packs. The ends projecting from the abdominal wound should be left fairly long, and those going to the pelvis or to the intestinal sutures should be indicated by a knot or a safety-pin, so as to avoid confusion later.

If the abdominal wound be very large, a suture or two may be applied at each end to somewhat diminish its size and to obviate the protrusion through the unhealed wound of a knuckle of gut, although this last complication has in no way interfered with recovery in the cases where it has occurred.

The dressing should be copious. Crumpled gauze should be applied next the wound in abundance, and over this many layers of flat gauze, the whole being retained in place by a bandage of Scultetus.

The patient is to be speedily returned to a well-warmed bed, not in the general ward, and be allowed to retain the same bed until fairly convalescent. The frequent moving of such patients is extremely undesirable. The foot of the bed may be raised twelve or eighteen inches, and the fluid will thus

readily drain out of the pelvis. Some surgeons prefer Fowler's position, with the head of the bed raised, so as to drain into the pelvis; but while good results have been obtained by both methods, the former commends itself to us as aiding in overcoming the shock, as well as making very efficient drainage.

Hypodermoclysis, or intravenous infusion of normal salt solution, may be begun now or continued if not completed as soon as the operation. When the patient comes out of ether a high enema of hot normal salt solution should be given, and may be repeated every two or three hours. It both allays the thirst and is a valuable adjunct to the abdominal drainage. A pint is readily absorbed by the colon in a short time.

After-treatment.—If the patient reacts from the perforation, he must be nourished, but all food by the mouth is to be avoided as long as possible. Six to eight ounces of milk peptonized for a half-hour, with some prepared beef-juice, may be given by enema every two or three hours, the balance of the pint being of the normal salt solution,—fortunately there is no palate in the lower bowel, and such mixtures are well tolerated. Hypodermatic stimulation is indicated as in other cases of similar nature. Hypodermoclysis of normal salt is likewise a valuable adjuvant, and may be repeated in quantities of a pint or two every four or six hours. Indeed, we think that the more fluid that can be absorbed from the lymphatics in these ways—hypodermatically and from the colon—the better it is for the patient; it overcomes the typhoid toxæmia, which is too often the precursor of death, and drains the peritoneal cavity from the mucous lining of the intestines outward. A few hours after the operation the outer dressings will probably be found saturated with the salt solution left in the abdomen at the time of operation, and will require changing. This saturation of the outer dressings may occur every four to six hours at first, being a favorable sign; the gauze packs acting as siphons and draining the peritoneal cavity in a most satisfactory manner.

Ice to the abdomen we regard as harmless, but of doubtful utility.

We have found in analyzing these cases that the greatest number of deaths took place between eight and twelve hours after the operation. Those patients who lived more than twelve hours after the operation had a fair chance of recovery, only 59.8 per cent. of them dying.

After twenty-four hours a teaspoonful of hot water may be given by the mouth every ten or fifteen minutes. Thus taken it is less apt to nauseate, and is probably all absorbed before reaching the stomach. No food should be given by the mouth until the third or fourth day at least, nutritive enemata being meanwhile continued. One or two cleansing enemata of plain water in the twenty-four hours are usually sufficient to remove all fæcal matter. When mouth-feeding is begun it should be borne in mind that the patient has both typhoid fever and a sutured area in his intestine, and he should be fed accordingly. Those cases do best where, the acme of the disease being past, a fairly liberal diet can be allowed early. Those patients, on the other hand, who, although they were in fairly good condition at the time of perforation and so have borne the operation well, but have yet several weeks of fever with which to contend, are very apt to die during the second or third week after the operation.

After the third or fourth day the surgeon should begin to think about removing the gauze drains; but let him be in no hurry about it; far better for them to remain a week or ten days than to be removed too soon. They should be well moistened with salt solution or hydrogen peroxide, and be given time to absorb it, and the utmost gentleness should be used in separating them from the intestines. It should be remembered that in time the peritoneal granulations will push the gauze away as a foreign body, and if the drains cannot be readily withdrawn on the first trial the attempt should be postponed for twelve or twenty-four hours. In drawing on the gauze it is well rather to try to separate the bowel from the gauze than the gauze from the gut. This is most expeditiously done by a sort of blunt dissection with the finger. Blind tugging at the drains is a most harmful as well as painful pro-

cedure, and it cannot be too often impressed upon the dresser that the gauze will come away of itself in time; it is only for the purpose of accelerating the cure and of keeping the wound fresh that nature should be thus assisted.

When the gauze has been removed it requires nice judgment to know how much to replace—this can only be learned by experience; if too much is replaced the healing of the sinus is delayed, and intestinal obstruction may be produced; if too little, a residual abscess may form in some pocket not efficiently drained; or rarely, where the adhesions are firm, intestinal obstruction may ensue from kinks of the bowel occurring from its sudden expansion on the removal of pressure.

A fæcal fistula may be regarded as of good prognostic import as far as life is concerned, as was mentioned before, and it has seldom failed to heal spontaneously, though, of course, delaying ultimate recovery. If it does not heal of itself it is better for the patient to endure the necessary discomfort until he is thoroughly convalescent, as a second laparotomy during typhoid fever is to be avoided if possible. If a second perforation occur, nevertheless, a second laparotomy must be performed and similar treatment instituted. The same remark applies to intestinal obstruction from bands or adhesions. Of eight cases where more than one operation has been performed, no fewer than three patients recovered (62.5 per cent. mortality), and, of these three, two endured three laparotomies.

EXPLORATORY LAPAROTOMIES.

Finally, a few words must be said about exploratory operations where the diagnosis is uncertain. Of twenty-six such operations where no peritoneal lesions were found, sixteen eventually recovered; only ten died, a mortality of 38.46 per cent. Of the nine fatal cases in which the duration of life after operation is known, only three died in less than twelve hours. Of these three, one (Finney) died from pulmonary embolism following iliac thrombosis; the second (J. F.

Mitchell) had had severe hæmatemesis and enterorrhagia shortly before operation, and was in a very precarious condition; while in the third case (Le Conte), which lived nearly seven hours after operation, the toxæmie state previously existing persisted without material change until death. In these three cases local anæsthesia was used, and in no way can the exploratory incision be held to have had any connection with the fatal termination.

This "laparotomie blanche," therefore, as it is named by Hagopoff, is practically never a cause of death, and, in our judgment, is to be recommended in all doubtful cases, especially as in some instances the irrigation of the peritoneal cavity employed has actually seemed to exert a beneficial influence on the course of the disease.

STATISTICAL SUMMARY.

Analysis of Whole Number of Cases.

Recovered	94
Died	208
Total	302
Mortality	74.03 per cent.

Analysis of Cases where Age and Sex are Known.

Age.	Male.			Female.			Total.			Mortality per Cent.		
	Recov.	Died.	Total.	Recov.	Died.	Total.	Recov.	Died.	Total.	Male.	Female.	Total.
Under 10 years.	3	2	5	1	3	4	4	5	9	40.0	75.0	55.5
10-15 "	9	10	19	3	3	6	12	13	25	52.6	50.0	52.0
15-20 "	6	29	35	1	2	3	7	31	38	83.0	66.6	81.8
20-30 "	16	70	86	8	19	27	24	89	113	81.4	70.3	78.0
30-40 "	13	41	57	6	6	12	19	50	69	77.2	50.0	72.1
40-50 "	8	10	18	1	2	3	9	12	21	55.5	66.6	57.1
50-60 "	0	3	3	0	1	1	0	4	4	100.0	100.0	100.0
Total	55	168	223	20	36	56	75	204	279	75.3	64.2	73.1

Analysis of Cases according to Sex Alone.

Sex.	Recovered.	Died.	Total.	Mortality.
Male	61	190	251	75.6 per cent.
Female	22	38	60	63.3 "

ANALYSIS ACCORDING TO DURATION OF PERFORATION BEFORE OPERATION.

Cases operated on.	Recovered.	Died.	Total.	Mortality.
First 12 hours after perforation..	35	95	130	73.0 per cent.
Second " " " ..	22	92	84	73.8 "
Third " " " ..	2	29	31	93.5 "
Over 30 hours " ..	18	37	55	97.2 "

ANALYSIS OF CASES WHERE DATE OF OPERATION IS KNOWN.

Year.	Recovered.	Died.	Total.	Mortality.
1884	1	0	1	0.0 per cent.
1885	0	2	2	100.0 "
1886	0	1	1	100.0 "
1887	0	3	3	100.0 "
1888	0	3	3	100.0 "
1889	1	3	4	75.0 "
1890	0	1	1	100.0 "
1891	1	5	6	83.3 "
1892	0	1	1	100.0 "
1893	0	4	4	100.0 "
1894	4	11	15	73.3 "
1895	5	12	17	70.5 "
1896	6	15	21	71.4 "
1897	3	9	12	75.0 "
1898	10	25	35	71.4 "
1899	6	40	46	86.9 "
1900	14	18	32	56.2 "
1901	11	15	26	57.6 "
1902	16	21	37	56.7 "
1903	4	21	25	84.0 "

ANALYSIS ACCORDING TO LUSTRUMS.

Period of time.	Recovered.	Died.	Total.	Mortality.
1884-1888	1	9	10	90.0 per cent.
1889-1893	2	14	10	87.5 "
1894-1898	28	72	100	72.0 "
1899-1903	51	115	199	90.2 "

STAGE OF DISEASE IN WHICH PERFORATION OCCURRED.

Perforation occurring.	Recovered.	Died.	Total.	Mortality.
First week of the disease.....	4	2	0	33.3 per cent.
Second " "	19	43	59	72.8 "
Third " "	22	81	103	78.6 "
Fourth " "	11	33	44	75.0 "
Fifth " "	5	26	25	80.6 "
Sixth " "	3	8	0	50.0 "
After sixth week of disease.....	4	12	19	75.6 "
In a relapse.....	8	7	15	49.9 "
In third relapse.....	0	1	1	100.0 "
In convalescence	7	4	11	80.9 "

TIME ELAPSING BETWEEN PERFORATION AND OPERATION, IN DETAIL.

Operation performed.	Recovered.	Died.	Total.	Mortality.
Within a half-hour	0	2	2	100.0 per cent.
" one hour	0	2	2	100.0 "
" two hours	2	4	0	00.0 "
" three hours	3	1	4	25.0 "
" four hours	2	12	14	85.7 "
" eight hours	17	30	56	00.6 "
" twelve hours	12	34	40	73.0 "
" eighteen hours	10	32	42	70.1 "
" twenty-four hours	12	30	42	71.4 "
" thirty-six hours	2	20	31	03.5 "
" forty-eight hours	4	10	20	80.0 "
" seventy-two hours	8	10	18	55.5 "
" five days	4	0	13	00.2 "
" two weeks	1	1	2	50.0 "
After two weeks	1	1	2	50.0 "

ANALYSIS AS TO THE PERFORATION.

	Recovered.	Died.	Total.	Mortality.
I. Number of perforation.				
Single	65	171	236	72.4 per cent.
Multiple	5	30	35	85.7 "
II. Size of perforation.				
Under ⅛ inch	35	37	72	51.3 "
" ½ "	17	56	73	76.7 "
Over ½ "	7	16	23	00.5 "
III. Site of perforation.				
Within 12 inches of cæcum	32	108	140	77.1 "
" 24 " "	7	32	39	82.0 "
" 36 " "	1	0	7	85.7 "
Over 3 feet from "	1	3	4	75.0 "
IV. Perforation of:				
Cæcum or ascending colon	1	4	5	80.0 "
Transverse colon	0	1	1	100.0 "
Sigmoid loop	0	1	1	100.0 "
Meckel's diverticulum	1	2	3	00.0 "
Appendix	4	4	8	50.0 "

TREATMENT OF GUT.

	Recovered.	Died.	Total.	Mortality.
Intestinal resection with end-to-end anastomosis	1	4	5	80.0 per cent.
Intestines evacuated through the perforation or a puncture	1	1	2	50.0 "
Enterotomy for tympany	1	5	0	83.3 "

DURATION OF AFTER-TREATMENT IN FATAL CASES.

Time.	Cases.	Per Cent. of Whole Number.
Died on table	5	2.1
" under 1 hour	11	4.8
" " 4 hours	20	8.8
" " 8 "	24	10.4
" " 12 "	20	12.6
" " 18 "	15	0.5
" " 24 "	18	7.8
" " 36 "	17	7.4
" " 48 "	21	0.1
" " 72 "	23	10.0
" " 5 days	21	0.1
" " 1 week	7	3.1
Lived over 1 "	11	4.8
" " 2 weeks	3	1.8
" 30 days or more	4	1.8
Total	220	100.0

ANALYSIS AS TO DRAINAGE.

Cases.*	Recovered.	Died.	Total.	Mortality.
Requiring drainage	72	180	258	72.0 per cent.
Not requiring drainage	11	18	29	62.0 "

NUMBER OF OPERATIONS PERFORMED.

Cases.	Recovered.	Died.	Total.	Mortality.
More than one operation performed,	3†	5	8	62.5 per cent.

CASES DEVELOPING A FAECAL FISTULA AFTER OPERATION.

Time.	Recovered.	Died.	Total.
Within 24 hours	2	1	3
" 36 "	1	0	1
" 48 "	1	0	1
" 72 "	1	0	1
" 5 days	3	1	4
" 1 week	1	0	1
Over 1 "	1	0	1
" 2 weeks	2	0	2
Time unknown	2	0	2
Total cases	14	2	10
Mortality per cent	12.5		

Treated by	Recovered.	Died.	Total.	Mortality.
Irrigation and drainage	40	130	170	73.8 per cent.
" but no drainage	7	15	22	68.0 "
No irrigation and no drainage	2	0	2	...
Wiping and drainage	12	19	31	61.0 "
" but no drainage	0	2	2	100.0 "
No wiping and no drainage	2	2	4	50.0 "
Drain, no wiping nor irrigation	10	7	17	41.0 "
Wiping, irrigation, and drainage	1	10	11	90.0 "
Eventration	3	8	11	72.0 "
False anus established	0	4	4	100.0 "

* Excluding cases where patient died on the table.

† Of the three patients who survived, two endured three operations.

	Recovered.	Died.	Total.	Mortality.
Ambulatory cases	7	10	23	00.5 per cent.
Relapse after operation	6	1	7	14.3 "
Two or more relapses after operation	2	0	2	0.0 "
With other complications	8	11	19	57.8 "
With a subsequent perforation	0	12	12	100.0 "
With a second perforation not found at operation	0	3	3	100.0 "
Perforation not found	5	15	20	75.0 "
Intestinal sutures not holding	13	9	22	40.9 "
Supposed to have recovered from a previous perforation	0	2	2	100.0 "
Subsequent protrusion of bowel through wound	2	0	2	0.0 "

ANALYSIS AS TO SITE OF INCISION.

Site.	Recovered.	Died.	Total.	Mortality.
Median hypogastric	21	75	00	78.12 per cent.
Right iliac	44	97	141	60.5 "
Left iliac, abscess pointing	2	0	2	
Median epigastric	1	3	4	75.0 "
Right hypochondriac	0	1	1	100.0 "
Multiple incisions	3	11	14	78.5 "
Drainage through loin, flank, or vagina	1	4	5	80.0 "

ANALYSIS OF CAUSE OF DEATH IN EIGHTY-NINE CASES IN WHICH IT IS GIVEN.

Causes of Death after Operation.	Under Twelve Hours.	Under Twenty-four Hours.	Under Three Days.	Under One Week.	Under Two Weeks.	Over Two Weeks.	Total.	Per Cent. of Whole Number.
Pre-existent peritonitis	17	6	16	4	1	0	44	49.4
Toxæmia of typhoid fever	2	0	5	2	0	0	9	10.1
Peritonitis from subsequent perforation	0	0	3	2	3	2	10	11.2
Exhaustion	0	0	0	1	3	1	5	5.6
Intestinal hæmorrhage	0	0	1	2	0	0	3	3.4
Intestinal obstruction	0	0	3	0	0	0	3	3.4
Other causes, uninfluenced by operation	7	0	8	0	0	0	15	16.8

ANALYSIS OF MISCELLANEOUS LAPAROTOMIES DURING TYPHOID FEVER.

Operations for	Recovered.	Died.	Total.	Mortality.
Appendicitis	12	4	10	25.0 per cent.
Disease of gall-bladder	4	8	12	60.0 "
Abscess of liver	1	1	2	50.0 "
Diseases of pelvic organs	5	0	5	——
Suppurating mesenteric glands	0	3	3	100.0 "
Intussusception	0	1	1	100.0 "
Chronic intestinal obstruction	1	0	1	——
Peritonitis of unknown cause	2	1	3	33.3 "

LIST OF CASES ANALYZED.

N.B.—References to Keen, from Nos. 1 to 83 inclusive, refer to the tables in "Surgical Complications and Sequels of Typhoid Fever," Philadelphia, 1898; from Nos. 84 to 158 inclusive, to the tables in "Surgical Treatment of Typhoid Fever," New York State Medical Association Transactions, 1899. References to Finney are to tables in Johns Hopkins Hospital Reports, vol. viii.

INTESTINAL PERFORATION.

No.	Operator.	Result.	Reference.
1.	Abbe.	Recov.	New York Med. Record, Jou. 6, 1895. Keen, 25. Finney, 1.
2.	"	Died.	Keen, Trans. New York State Med. Assoc., 1899. Keen, 85.
3.	"	"	Ibid. Keen, 84.
4.	Alexnodroff.	"	Journ. de Clin. et Thérapeut. Bofontilles, Paris, 1804, ii. 735. Loctop, Khirurg. Obshtsh. v. Mosk., 1801, x. 121-120, in ANNALS OF SURGERY, 1807, i. 207. Keen, 27.
5.	Allen, D. P.	"	American Journ. Med. Sciences, January, 1902, p. 43.
6.	"	"	Ibid., p. 50.
7.	Allingham.	"	Trans. Clin. Soc. London, vol. xxvii. Brit. Med. Journ., 1894, vol. i. p. 578. Keen, 24. Finney, 3.
8.	Anderson.	Recov.	Brit. Med. Journ., July 23, 1868. Keen, 86.
9.	Andrews.	"	ANNALS OF SURGERY, 1902, vol. xxxvi, p. 623.
10.	Armstrong.	Died.	Montreal Medical Journal, Feb., 1807, p. 001. Keen, 52. Finney, 4.
11.	"	"	Brit. Med. Journ., 1890, vol. ii. p. 1021. Keen, 53.
12.	"	"	Ibid. Keen, 54.
13.	"	"	Ibid. Keen, 55.
14.	" (Reporter).	"	Ibid. Keen, 56.
15.	" "	"	Ibid. Keen, 57.
16.	" "	"	Ibid. Keen, 58.
17.	"	"	Keen, 79.
18.	Audet.	"	Archives do Méd. et de Pharmacie Milit., 1800, xxxiv., p. 134.
19.	"	"	Ibid.
20.	Auvray.	"	Bull. et Mém. de la Soc. Anat. de Paris, jan., 1901, p. 05.
21.	"	"	Ibid., p. 06.
22.	Balch.	"	Trans. Amer. Surg. Assoc., 1900, p. 419, Case 24.
23.	Ballance.	"	Cargill, Brit. Med. Journ., Dec. 15, 1900, p. 1738.
24.	Banzet.	"	F. Junqua, Thèse de Paris, 1901, p. 90.
25.	Bartleet.	"	Medical News, November, 1887.
26.	Beach.	"	Trans. Amer. Surg. Assoc., 1900, p. 410, Case 0. Boston Med. and Surg. Journ., Oct., 1898, p. 300. Keen, 87. (Age given here as fifteen years.)
27.	Beckett.	"	South. California Practitioner, 1899, xiv., 111. Keen, 88.
28.	Bell, James.	"	Medical Chronicle, September, 1895, p. 401. Keen, 18.
29.	"	"	Ibid. Keen, 21.

No.	Operator.	Result.	Reference.
30.	Berg.	Recov.	New York Medical Record, March 23, 1901, p. 441.
31.	Bigger.	Died.	British Medical Journal, 1896, vol. i. p. 86. Keen, 86.
32.	Blake.	"	Bost. Med. and Surg. Journ., Feb. 5, 1903, p. 140. Case 10.
33.	Bland-Sutton.	"	Trans. Clin. Soc. London, vol. xxvii. Brit. Med. Journ., 1894, vol. i. p. 578. Keen, 23. Finney, 90.
34.	Bocaloglu.	"	Mauger, Thèse de Paris, 1900, p. 101.
35.	Bogart.	"	ANNALS OF SURGERY, May, 1899. Keen, 44.
36.	Boinet.	"	Archives Gén. de Méd., October, 1899, p. 426. Mauger, Thèse de Paris, 1900, p. 199.
37.	" (Reporter.)	"	Archives Gén. de Méd., 1866, p. 542.
38.	" "	"	Ibid., p. 543.
39.	Bontecou.	"	Journ. Amer. Med. Assoc., Jan. 28, 1888, p. 106. Keen, 5.
40.	"	"	Ibid., March 20, 1899, p. 455. Keen, 11.
41.	Bowlby.	Recov.	Lancet, January 30, 1897, p. 312. Proc. Royal Med. and Chir. Soc. of London, vol. ix. Keen, 61.
42.	"	"	Lancet, January 16, 1903.
43.	Briddon.	Died.	ANNALS OF SURGERY, February, 1896, p. 198. Keen, 43.
44.	Briggs, C. E.	Recov.	Amer. Jour. Med. Sciences, January, 1902, p. 47.
45.	Brooks.	"	Trans. Amer. Surg. Assoc., 1900, p. 415. Case 17.
46.	Bruce.	"	Canada Lancet, March, 1902, abstracted in Amer. Med., May, 1902, p. 747.
47.	Brown, F. Tilden.	Died.	ANNALS OF SURGERY, March, 1903, p. 380.
48.	"	"	Ibid.
49.	"	Recov.	Ibid.
50.	Brun.	Died.	Bull. et Mém. de la Soc. de Chir. de Paris, nov. 25, 1896, p. 731. Keen, 66.
51.	Burrell.	"	Trans. Amer. Surg. Assoc., 1900, p. 417. Case 19.
52.	Blake, Jos. A.	"	Personal communication.
53.	"	Recov.	Ibid.
54.	Cameron.	Died.	Philadelphia Med. Journ., March 3, 1900, p. 520.
55.	Canoli.	"	Bull. de Soc. Lancisiana d. Osp. di Roma (1895), 1896, xv. i., 9-14. Quoted from Keen, 32.
56.	"	"	Ibid. Keen, 33.
57.	Cargill (Reporter).	"	Brit. Med. Journ., December 18, 1999, p. 1738.
58.	" "	"	Ibid.
59.	Celos. "	"	Bull. et Mém. de la Soc. Anat. de Paris, mai, 1899, p. 503.
60.	Champlin.	Recov.	The Plexus, Chicago, 1896, No. 5, vol. v. p. 134. Keen, 155.
61.	"	Died.	Ibid. Keen, 159.
62.	"	"	Ibid. Keen, 157.
63.	Chevalier.	Recov.	Bull. et Mém. de la Soc. de Chir. de Paris, juin 11, 1902, p. 902.
64.	"	"	Ibid., p. 903.
65.	Cholzow.	Died.	Ann. der Russ. Chir., 1899, Heft 2, in Centralbl. f. Chir., 1900, No. 42. Keen, 50.

No.	Operator.	Result.	Reference.
90.	Cushing, H.	Recov.	Johns Hopkins Hosp. Bull., 1898, vol. ix. p. 207. Ibid. Reports, vol. viii. p. 219. Keen, 99.
97.	"	Died.	Johns Hopkins Hosp. Reports, vol. viii. p. 218. Keen, 01.
98.	"	"	Ibid. Keen, 02.
99.	"	"	Phila. Med. Journ., March 3, 1900, p. 595. Keen, 03.
70.	"	Recov.	Annals of Surgery, 1901, vol. xxxiii. p. 550.
71.	Dalton.	Died.	Medical Review, St. Louis, 1898, vol. xxxviii. p. 302. Keen, 05.
72.	"	"	Ibid. Keen, 99.
73.	Dalziel.	"	Keen, Trans. New York State Med. Assoc. Keen, 97.
74.	"	"	Ibid. Keen, 98.
75.	"	"	Ibid. Keen, 99.
76.	"	"	Ibid. Keen, 100. Flancy, 34.
77.	"	"	Ibid. Keen, 101.
78.	"	Recov.	Ibid. Keen, 102.
79.	Dandridge.	"	Keen, 28.
80.	"	Died.	Cincinnati Lancet-Clinic, Aug. 21, 1897, p. 177. Keen, 05.
81.	Da Costa.*	"	Allyn, Phila. Med. Journ., Aug. 3, 1901, p. 193. "Modern Surgery," 1903, 4th ed., p. 724.
82.	Davis, G. G.	Recov.	Univ. Med. Mag., May, 1900, p. 172. Episcopal Hospital Records, Philadelphia.
83.	"	Died.	Univ. Med. Mag., loc. cit., p. 173. Episcopal Hospital Records.
84.	"	"	Episcopal Hospital Records.
85.	"	"	Ibid.
86.	"	"	Ibid.
87.	"	"	Ibid.
88.	"	"	Ibid.
89.	Davis, R. T.	Recov.	Amer. Med., January 18, 1902, p. 116.
00.	Dennesly & Malet.	"	Lancet, May 25, 1901, p. 1496.
01.	Denver, H. C.	Died.	Episcopal Hospital Records.
02.	"	"	Amer. Journ. Med. Sci., Feb., 1898, p. 191. Keen, 104.
03.	"	"	Episcopal Hospital Records.
04.	"	"	Ibid.
05.	"	"	Ibid.
99.	Denver, J. H.	Recov.	Amer. Journ. Med. Sci., Feb., 1898, p. 191. Keen, 103.
07.	"	Died.	Annals of Surgery, 1898, p. 144. Keen, 105.
08.	Delore.	"	Mouriquand, Lyon Méd., juillet 12, 1903, p. 41.
09.	"	"	Ibid., p. 40.
100.	Depage.	Recov.	Journ. de Chir. et Annales de la Soc. Belge de Chir., November-December, 1902.
101.	Elliot.	"	Trans. Amer. Surg. Assoc., 1900, p. 415. Case 16. New York Med. Record, Dec. 22, 1900, p. 998. Keen, 94.
102.	Ferraresi.	"	Bull. de Soc. Lancisiana d. Osp. di Roma (1895), 1899, xv. I., 9-14 (quoted in Keen). Keen, 31.

* See also Nos. 300, 301, and 302.

No.	Operator.	Result.	Reference.
103.	Ferrier.	Died.	Bull. et Mém. de la Soc. Méd. des Hôpitaux, fev. 8, 1001, p. 112.
104.	"	"	Ibid., p. 108.
105.	Finney.	"	ANNALS OF SURGERY, 1807, p. 233. Johns Hopkins Hospital Reports, vol. viii. p. 187. Keen, 00.
100.	"	"	Ibid., loc. cit. Keen, 07.
107.	"	Recov.	Ibid., loc. cit., p. 180. Keen, 08.
108.	"	Died.	Johns Hopkins Hosp. Reports, vol. viii. p. 187. Keen, 100.
100.	"	Recov.	Ibid., p. 188. Keen, 107.
110.	"	Died.	Osler, Phila. Med. Journ., Jan. 10, 1001, p. 110. Johns Hopkins Hospital Reports, vol. x., No. 8, p. 130, Case 17.
111.	"	"	Johns Hopkins Hospital Reports, loc. cit., Case 14, p. 120.
112.	Gerster.	Recov.	Berg, New York Med. Record, March 23, 1001, p. 443.
113.	Gessclewitch and Dombrowski.	Died.	Loitop. Russk. Chir., 1807, vol. ii. p. 407. (Quoted in Keen.) Keen, 00. Finney, 46.
114.	Gessclewitch and Wonoch.	"	Ibid. Keen, 70. Finney, 50.
115.	Gessclewitch and Kadjanov.	"	Ibid. Keen, 71. Finney, 49.
116.	Gessclewitch and Wanoch.	"	Ibid. Keen, 72. Finney, 48.
117.	Gessclewitch and Wanoch.	"	Ibid. Keen, 73. Finney, 47.
118.	Gibbon.	Recov.	Pennsylvania Hospital Records.
119.	"	Died.	Ibid.
120.	"	"	Personal communication. St. Joseph's Hosp. Records, Philadelphia.
121.	"	"	Ibid. Polyclinic Hospital Records, Philadelphia.
122.	"	"	Pennsylvania Hospital Records.
123.	"	"	Ibid.
124.	"	"	Ibid.
125.	Godwin.	"	Thornton and Godwin, Lancet, August 17, 1001.
126.	Goodal & Richards.	"	British Med. Journal, 1808, vol. i. p. 1320. Keen, 130.
127.	Gosset.	"	Bull. et Mém. de la Soc. de Chir. de Paris, dec. 20, 1000, p. 110.
128.	Gray.	"	New York Med. Record, April 22, 1800, p. 507. Case 0. Keen, 108.
129.	Guinon (Reporter).	"	Revue Mensuelle des Maladies de l'Enfance, juillet, 1800, p. 200.
130.	Hagopoff.	Recov.	Bull. et Mém. de la Soc. de Chir. de Paris, juin 18, 1002, p. 080.
131.	Haggard.	Died.	Trans. South. Surg. and Gyn. Congr., 1800, p. 148.
132.	Hahn.	"	Frank, Beilage zum Centralbl. f. Chir., 1888, vol. xxiv. p. 51. Keen, 0. Finney, 51.
133.	"	"	Ibid. Keen, 10. Finney, 52.
134.	Hare.	"	Intercolonial Quarterly Journ., Feb., 1805. Keen, 20.
135.	Harrison, V. W.	"	North Carolina Med. Journ., Dec. 5, 1807, p. 308. Keen, 82. Finney, 50.
130.	Harrisson.	"	Brit. Med. Journ., Oct. 20, 1804. Keen, 84.

No.	Operator.	Result.	Reference.
137.	Harte.	Died.	Pennsylvania Hospital Records.
138.	"	"	Ibid.
139.	"	"	Ibid.
140.	"	Recov.	Episcopal Hospital Records.
141.	"	Died.	Pennsylvania Hospital Records.
142.	"	"	Ibid.
143.	"	"	Episcopal Hospital Records.
144.	"	"	Pennsylvania Hospital Records.
145.	"	Recov.	Ibid.
146.	"	"	Ibid.
147.	"	Died.	Ibid.
148.	"	"	Ibid.
149.	"	"	Ibid.
150.	"	"	Ibid.
151.	"	"	Ibid.
152.	"	"	Ibid.
153.	"	"	Ibid.
154.	"	Recov.	Ibid.
155.	"	Died.	Ibid.
156.	"	"	Ibid.
157.	Hayes.	"	Amer. Med., Sept. 6, 1902, p. 379.
158.	"	Recov.	Ibid.
159.	"	Died.	Ibid.
160.	"	Recov.	Ibid.
161.	"	"	Ibid.
162.	"	Died.	Ibid., p. 389.
163.	"	"	Ibid.
164.	Hearn.	"	Ibid., May 2, 1903, p. 799.
165.	Henneld.	"	Med. Rev., St. Louis, 1898, vol. xxxvii, p. 302. Keen, 109.
166.	Heurtreaux and Waquet.	Recov.	Mauger, Thèse de Paris, 1900, p. 112.
167.	Heuston.	"	Brit. Med. Journ., November 10, 1901.
168.	Hill, W.	"	Keen, 49.
169.	Hollis.	Died.	Lancet, 1899, vol. I. p. 1284. Keen, 50.
170.	Hotchkiss.	"	N. Y. Med. Journ., Jan. 11, 1896. Keen, 42. Finney, 59.
171.	Hutchinson, J. P.	Recov.	Pennsylvania Hospital Records.
172.	"	Died.	Children's Hospital Records, Philadelphia.
173.	"	"	Episcopal Hospital Records.
174.	"	"	Ibid.
175.	"	"	Lloyd and Coley, Phila. Med. Journ., Jan., 1903, p. 133 (Methodist Hospital Records, Philadelphia).
176.	"	Recov.	Episcopal Hospital Records.
177.	"	Died.	Pennsylvania Hospital Records.
178.	"	Recov.	Ibid.
179.	Hutchison, J. A.	Died.	Keen, 77. Finney, 60. Mauger, 70.
180.	"	"	Keen, 78.
181.	Ill, C. L.	Recov.	Petry, Phila. Med. Journ., Dec. 13, 1902, p. 936.
182.	Jackson.	Died.	N. Y. Med. Record, Oct. 7, 1899, p. 519. Keen, 119.
183.	Jones.	Recov.	Annals of Surgery, July, 1901.
184.	Kadjan.	"	Gesselewitch, St. Petersb. med. Woch., 1898, n. f. 15, No. 3, s. 21.
185.	Kammerer.	"	Seibert, Archives of Pædiatrics, Sept., 1902.
186.	Kholizoff.	Died.	La Presse Méd., mai 18, 1898, p. 271.

No.	Operator.	Result.	Reference.
187.	Kimura.	Died.	Sei-I-Kwai Med. Journ., 1899, vol. ix. p. 55, quoted in Brit. Med. Journ., 1899, vol. II. p. 777. Keen, 14.
188.	Kingsley.	"	Annals of Surgery, Mar., 1897, p. 233. Keen, 39. Finney, 93.
189.	Kirkpatrick.	"	Keen, 89.
190.	Korte.	"	Archiv f. klin. Chir., 1892, vol. xliv. p. 646. Keen, 10. Finney, 65.
191.	Kropowski.	Recov.	Gesselewitch, St. Petersb. med. Woch., 1898, n. f. 15. No. 3, s. 23. Keen, 111.
192.	Laidley.	Died.	Amer. Journ. of Obst., Nov., 1895, p. 701. Keen, 38.
193.	Lapinec.	"	Tyson, Trans. Coll. Phys. Phila., 1902, p. 133. Personal communication from Prof. Tyson.
194.	Le Conte.*	Recov.	Annals of Surgery, 1901, vol. xxxiii. p. 645.
195.	"	Died.	Pennsylvania Hospital Records.
196.	"	"	Ibid.
197.	"	"	Ibid.
198.	Legueu.	"	Mauger, Thèse de Paris, 1900, p. 107.
199.	"	"	Bull. et Mém. de la Soc. de Chir. de Paris, dec. 20, 1900, p. 1159.
200.	"	Recov.	Ibid., loc. cit., p. 1157. Lolson, Revue de Chir, 1901, vol. xxiii. p. 170.
201.	Lejars.	Died.	Bull. et Mém. de la Soc. de Chir. de Paris, nov., 1895. Keen, 40. Finney, 68.
202.	"	"	Ibid., nov. 25, 1899. Keen, 70. Finney, 99.
203.	"	"	Ibid., dec. 20, 1900, p. 1158. Mauger, Thèse de Paris 1900, p. 97.
204.	Levison.	"	Voorsanger, Amer. Med., Aug. 22, 1903, p. 318.
205.	Lolson.	Recov.	Bull. et Mém. de la Soc. de Chir. de Paris, dec. 5, 1900. Bull. et Mém. de la Soc. Méd. des Hôplt. de Paris, fevrier 8, 1901, p. 107. Revue de Chir., 1901, vol. xxiii. p. 181.
206.	Lothrop.	Died.	Munro, Bost. Med. and Surg. Journ., Feb. 6, 1903. Case 11.
207.	Lücke.	"	Deut. Zeits. f. Chir., 1889-1857, Bd. xxv. pp. 1-4. Med. News, Nov., 1887. Trans. Amer. Surg. Assoc., 1888, p. 422.
208.	Lund.	"	Warren, Trans. Amer. Surg. Assoc., 1900, p. 414. Case 14. Keen, 112. Finney, 70.
209.	"	"	Ibid., p. 493, Case 2.
210.	"	"	Ibid., p. 498. Bost. Med. and Surg. Journ., 1900, vol. I. p. 988, Case 5.
211.	Lutz.	Recov.	Med. Review, St. Louis, 1898, vol. xxxvii. 392. Keen, 113.
212.	"	"	Ibid. Keen, 114.
213.	"	Died.	Ibid. Keen, 115.
214.	Margarucci.	"	Bull. di Soc. Lancisiana d. Osp. di Roma, 1898, xviii. 310. Quoted by Keen. Keen, 116. Finney, 71.
215.	Marsden.	Recov.	Lancet, June 23, 1900.
216.	"	Died.	Ibid.

* Another patient operated on at the Pennsylvania Hospital by Dr. Le Conte is convalescent, six weeks having elapsed since operation.

No.	Operator.	Result.	Reference.
217.	Martin, Edw.	Died.	Univ. Med. Mag., June, 1890, p. 502. Keen, 117.
218.	"	Recov.	Ibid. Keen, 118.
219.	McArthur.	Died.	Annals of Surgery, 1902, vol. xxxvi. p. 624.
220.	"	"	Ibid.
221.	"	"	Ibid.
222.	McReynolds.	Recov.	Trans. Coll. Phys. Phila., 1902, p. 134. Personal communication.
223.	"	Died.	Proceedings of Phila. Acad. of Surgery, April 6, 1903. Personal communication.
224.	"	"	Ibid.
225.	"	"	Ibid.
226.	Mielescu.	Recov.	Therapeutische Monatshefte, Dec., 1902, p. 631.
227.	Mignon.	Died.	Loison, Revue de Chir., Paris, 1901, vol. xxiii. p. 170.
228.	Mikulicz.	Recov.	Volkmann's Samml. klin. Vortrilge, No. 202. Chirurgie, April, 1884, No. 83. Keen, 1. Finney, 72.
229.	"	Died.	Verhandlungen der Deutschen Gesellschaft f. Chir., xvIII Kongress, s. 324. Keen, 7. Finney, 74.
230.	Mitchell, C. F.	"	Pennsylvania Hospital Records.
231.	"	"	Ibid.
232.	"	Recov.	Ibid.
233.	Mitchell, J. F.	"	Johns Hopkins Hosp. Reports, vol. x. p. 403. Case 11.
234.	"	Died.	Ibid., p. 412. Case 12.
235.	"	Recov.	Ibid., p. 410. Case 13. Osler, Philo. Med. Journ., Jan. 19, 1901, p. 110.
236.	"	Died.	Johns Hopkins Hosp. Reports, vol. x. p. 423. Case 15.
237.	"	"	Ibid., p. 426. Case 19.
238.	Mixter.	"	Trans. Amer. Surg. Assoc., 1900, p. 410. Case 8.
239.	Monks.	"	Munro, Boston Med. and Surg. Journ., February 5, 1903, p. 149. Case 15.
240.	Monod.	"	Bull. et Mém. de la Soc. de Chir. de Paris, nov. 18, 1899. Keen, 92.
241.	"	"	Ibid., dec. 12, 1900.
242.	Moore, J. E.	"	Northwestern Lancet, 1898, vol. xviii. p. 135. Keen, 110.
243.	Morestin,	"	Mauger, Thèse de Paris, 1900, p. 104.
244.	Morton, T. G.	"	Medical News, Dec. 24, 1887, p. 730. Keen, 9.
245.	Mower-White.	Recov.	Lancet, January 26, 1901.
246.	Moynihan.	Died.	British Med. Journ., 1899, vol. i. p. 1097. Keen, 120.
247.	Munro.	"	Burrell and Bottomly, Boston City Hospital Reports, 1898, p. 120. Keen, 122. Finney, 78.
248.	"	"	Trans. Amer. Surg. Assoc., 1900, p. 400. Case 7. Bost. Med. and Surg. Journ., Feb. 5, 1903. Case 9. Keen, 121.
249.	"	"	Trans. Amer. Surg. Assoc., 1900, p. 400. Case 6. Bost. Med. and Surg. Journ., 1900, vol. i. p. 388. Case 6. Ibid., Feb. 5, 1903, p. 149. Case 9.
250.	"	"	Trans. Amer. Surg. Assoc., loc. cit. Case 12. Bost. Med. and Surg. Journ., loc. cit., 1903. Case 7.

No.	Operator.	Result.	Reference.
251.	Munro.	Died.	Trans. Amer. Surg. Assoc., loc. cit. Case 20. Bost. Med. and Surg. Journ., loc. cit. Case 8.
252.	"	"	Bost. Med. and Surg. Journ., loc. cit., p. 148. Case 1.
253.	"	"	Ibid. Case 2.
254.	"	"	Ibid. Case 3.
255.	"	"	Ibid. Case 4.
256.	"	"	Ibid. Case 5.
257.	Murphy.	"	Keen, 22.
258.	"	Recov.	Keen, 41.
259.	"	"	Journ. Amer. Med. Assoc., April 11, 1903, p. 978.
260.	Mübsom.	"	Deutsche med. Woch., 1901, No. 32, s. 534.
261.	Müller.	"	Deutsche Militärärztliche Zeitsch., 1901, vol. xxx. s. 501.
262.	Neilson.	Died.	Episcopal Hospitol Records.
263.	"	"	Ibid.
264.	Nevlsou.	"	Amer. Journ. Med. Sciences, Jan., 1902, p. 30. Case 1.
265.	"	"	Ibid., p. 41. Case 2.
266.	Niebols.	"	Bost. Med. and Surg. Journ., Feb. 5, 1903. Case 12.
267.	Nutt.	Recov.	McCormick, Therapeutic Gazette, Aug. 15, 1903, p. 513.
268.	Ortmonn.	Died.	Mikulicz, Verhandlungen der Deut. Gesellschaft f. Chir., xviii Kongress, p. 324, Keen, 7. Finney, 74.
269.	Panton.	Recov.	ANNALS OF SURGERY, Aug., 1897, p. 210. Keen, 54.
270.	Parkin.	Died.	British Med. Journ., 1895, vol. i. p. 102. Keen, 37.
271.	Patteson.	"	Taylor, Dublin Journ. Med. Sci., Jan., 1901, p. 3.
272.	Pearson.	Recov.	British Med. Journ., vol. i. p. 1007. Keen, 125.
273.	Peyrot.	"	Mauger, Thèse de Paris, 1900, p. 25.
274.	"	"	Ibid., p. 98.
275.	Pick.	Died.	Trans. Clin. Soc. London, 1898, p. 234. Brit. Med. Journ., 1898, vol. i. p. 1328. Keen, 120.
276.	Platt, J. E.	Recov.	Lancet, Feb. 25, 1899, p. 505. British Med. Journ., 1899, vol. i. p. 345. Keen, 127.
277.	"	Died.	Ibid. Keen, 128.
278.	"	"	Ibid. Keen, 129.
279.	"	"	Keen, 130.
280.	"	"	Keen, 131.
281.	Pluyette.	"	Mauger, Thèse de Paris, 1900, p. 105. Archives Générales de Médecine, 1899, N. S., vol. ii. p. 530.
282.	Porter.	"	Boston Med. and Surg. Journ., April 15, 1897, p. 354. Trans. Amer. Surg. Assoc., 1900, p. 411. Case 10. Keen, 93 and 132. Finney, 87.
283.	"	"	Trans. Amer. Surg. Assoc., 1900, p. 413. Case 13.
284.	Post.	"	Munro, Bost. Med. and Surg. Journ., Feb. 5, 1903. Case 14.
285.	Powers.	"	Keen, 134.

No.	Operator.	Result.	Reference.
286.	Price, Jos.	Recov.	Canada Lancet, April, 1898, p. 389. Med. and Surg. Reporter, 1899, p. 577. Keen, 49. Finney, 88.
287.	"	"	Canada Lancet, 1897, vol. xxx. p. 385. Med. and Surg. Reporter, loc. cit. Keen, 135.
288.	"	"	Canada Lancet, April, 1898. Med. and Surg. Reporter, loc. cit. Keen, 45. Finney, 89.
289.	Ruth.	Died.	Records of St. Timothy's Hospital, Roxborough, Phila.
290.	"	"	Ibid.
291.	"	"	Ibid.
292.	Renrd.	Recov.	Bull. et Mém. de la Soc. de Chir. de Paris, July 18, 1902, p. 980.
293.	Ricketts.	Died.	Cincinnati Lancet-Clinic, April 9, 1895, p. 383. Keen, 30.
294.	Rochard.	"	Bull. et Mém. de la Soc. de Chir. de Paris, dec. 20, 1899, p. 1191.
295.	"	"	Ibid.
296.	Rodman.	Recov.	Amer. Med., Nov. 23, 1901.
297.	Rogers.	Died.	Trans. South. Surg. and Gyn. Congress, 1890, p. 148.
298.	Ross, G. G.	"	Philadelphia Med. Journ., May 2, 1903, p. 748.
299.	Routier.	"	Bull. et Mém. de la Soc. de Chir. de Paris, dec. 20, 1899, p. 1191. Ibid., nov. 18, 1896, p. 728. Keen, 51.
300.	Ryan.	"	Australasian Medical Gazette, 1899, vol. xviii. p. 334. Keen, 137.
301.	Sacquépée.	"	Bull. et Mém. de la Soc. Anat. de Paris, 1899, vol. lxxiv. p. 443. Mauger, Thèse de Paris, 1900, p. 90.
302.	Saleeby.	Recov.	Phila. Med. Journ., Aug. 5, 1899, p. 270. Keen, 158.
303.	Senn.	Died.	Medical News, June 8, 1889, p. 622. Keen, 12. Finney, 93.
304.	"	"	Keen, 130.
305.	"	Recov.	Keen, 149.
306.	"	Died.	Keen, 141.
307.	Shepherd, F. J.	Recov.	Edinburgh Med. Journ., 1902, vol. liv. p. 531.
308.	"	"	Ibid., p. 532.
309.	"	"	Ibid., p. 534.
310.	Shoemaker, G. E.	Died.	Phila. Med. Journ., May 31, 1902, p. 981. Trans. Coll. Phys. Phila., 1902, p. 127.
311.	Sicur.	"	Loison, Revue de Chir., Paris, 1901, vol. xxiii. p. 179.
312.	Sifton.	Recov.	Chicago Clin. Review, April, 1895. Keen, 48. Finney, 94.
313.	Souligoux.	Died.	Mauger, Thèse de Paris, 1900, p. 102.
314.	"	"	Ibid., p. 103.
315.	Stewart, F. T.	"	Pennsylvania Hospital Records.
316.	"	"	Ibid.
317.	"	"	Ibid.
318.	"	Recov.	Ibid.
319.	"	Died.	Personal communication. Records of Polyclinic Hospital, Philadelphia.
320.	"	"	Ibid. Records of Jefferson College Hospital, Phila.
321.	Spellissy.	Recov.	Proceedings of Phila. Acad. of Surg., April 9, 1903

No.	Operator.	Result.	Reference.
322.	Surmay.	Died.	L'Union Méd., 1835, vol. xl. p. 001. Keen, 3.
323.	Taylor, H. M.	"	Virginia Med. Semi-monthly, Dec. 10, 1807, p. 516. Trans. South. Surg. and Gyn. Congr., 1800, p. 140. Case 1. Keen, 83.
324.	"	Recov.	Virginia Med. Semi-monthly, 1808-1800, vol. iii. p. 710. Trans. South. Surg. and Gyn. Congr., loc. cit., p. 142. Case 3. Keen, 142.
325.	"	Died.	Virginia Med. Semi-monthly, loc. cit. Trans. South. Surg. and Gyn. Congr., loc. cit., p. 142. Case 2. Keen, 143. Finney, 08.
320.	"	"	Maryland Med. Journ., 1809, vol. xiii. p. 101. Trans. South. Surg. and Gyn. Congr., loc. cit., p. 143. Case 4. Keen, 144.
327.	"	"	Trans. South. Surg. and Gyn. Congr., loc. cit.
328.	Taylor, W.	"	Dublin Journ. Med. Science, Jan., 1901, p. 1.
320.	Taylor, W. J.	"	Trans. Coll. Phys. Phila., 1800, p. 107. Keen, 145.
330.	"	"	Ibid., p. 108. Keen, 146.
331.	Thermet.	"	Monod & Vanverts, Revue de Chir., 1807, vol. xvii. p. 100, where it is quoted from Barbe, Thèse de Paris, Obs. 20.
332.	Thompson.	"	Trans. Amer. Surg. Assoc., 1900, p. 412. Case 11. Keen, 133.
333.	Thompson, J. E.	"	Trans. Texas State Med. Assoc., 1803, p. 200. Med. Chron., 1805, p. 401. Keen, 20. Finney, 101.
334.	"	"	Med. Chron., loc. cit. Keen, 30. Finney, 100.
335.	Thorndike.	"	Trans. Amer. Surg. Assoc., 1900, p. 400. Case 1.
336.	"	"	Ibid., p. 410. Case 18.
337.	"	Recov.	Boston Med. and Surg. Journ., Feb. 5, 1003. Case 13.
338.	Thurston.	"	Lancet, Oct. 14, 1800. Ibid., Feb. 1800, p. 1004. Keen, 118.
330.	Tiffany.	Died.	Keen, 147. Finney, 102.
340.	Trojanoff.	Recov.	Bolnitschnaia Gaseta Botkina, No. 23, 1804, abstracted in Med. News, 1804, vol. lxv. p. 600. Janqua, Thèse de Paris, 1001. Case 01. Keen, 20, 124.
341.	"	Died.	Lallop. russk. Chir., 1807, vol. ii. p. 277, in Manger, Thèse de Paris, 1000. Case 10. Keen, 74 and 123. Finney, 103.
342.	Vnlence.	"	Loison, Revue de Chir., Paris, 1901, vol. xxiii. p. 170.
343.	Van Duyn.	"	Keen, 140.
344.	Van Hook.	Recov.	Med. News, Nov. 21, 1891, p. 591. Keen, 15. Finney, 104.
345.	"	Died.	Ibid. Keen, 10. Finney, 105.
340.	"	"	Ibid. Keen, 17. Finney, 100.
347.	Wagner.	Recov.	Hellage z. Centralbl. f. Chir., 1880, No. 20, p. 00. Keen, 13. Finney, 107.
348.	Wanach.	Died.	Gesselewitch, St. Petersburg med. Woch., 1808, iii. n. f. 15, 23.
340.	Watson, F. S.	Recov.	Boston City Hosp. Reports, 1808, p. 127. Bost. Med. and Surg. Journ., 1800, No. 13. Ibid., 1000, vol. i. p. 088. Case 4. Trans. Amer. Surg. Assoc., 1000, p. 407. Case 4. Keen, 47.

No.	Operator.	Result.	Reference.
350.	Watson, F. S.	Died.	Trans. Amer. Surg. Assoc., 1900, p. 414. Case 15.
351.	Warren, J. C.	"	Ibid., p. 407. Case 3.
352.	Weir.	"	ANNALS OF SURGERY, Dec., 1897. Keen, 81. Finney, 110.
353.	Willard.	"	Ibid., 1800, vol. xxix. p. 503. Keen, 151.
354.	v. Winiwarter.	"	Polls, Annal. de la Soc. Méd. Chir. de Liege, 1807, vol. xxxvi. p. 203. Keen, 152. Finney, 80.
355.	Wladisslew.	Recov.	Gesselewitch, St. Petersburg med. Woch., 1898, vol. iii., n. f. 15, 23. Keen, 153.
356.	Woodward.	Died.	Boston Med. and Surg. Journ., 1808, vol. cxxxix. p. 544. Keen, 154.
357.	Yule.	Recov.	Edinburgh Med. Journ, 1800, vol. xlvii. p. 300.
358.	"	Died.	Ibid., p. 361.
359.	Zeidler.	"	Netschajeff, Bolnitsch. Gas. Botk., 1804, p. 569; in Keen, 75.
300.	Da Costa.	"	"Modern Surgery," 4th ed., 1003, p. 724.
361.	"	"	Ibid.
362.	"	"	Ibid.

Mackenzie has reported (Lancet, September 20, 1003, p. 807) two recoveries after operation by Battle for typhoid perforation. These are not included in the above tables.

EXPLORATORY LAPAROTOMIES.

1.	Armstrong.	Recov.	Lafleur, Montreal Med. Journ., Feb., 1001, p. 80.
2.	Auvray.	Died.	Bull. et Mém. de la Soc. Anat. de Paris, jan., 1001, p. 08.
3.	Berg.	Recov.	New York Med. Record, March 23, 1001, p. 443.
4.	Briggs.	"	Amer. Journ. Med. Sciences, Jan., 1902, p. 45.
5.	Bull.	"	New York Med. Record, June 1, 1001, p. 873.
0.	Cushing, H.	"	Johns Hopkins Hospital Reports, vol. viii. p. 220.
7.	"	Died.	Philadelphia Med. Journ., March 3, 1000, p. 507.
8.	Davis, G. G.	Recov.	Univ. Med. Mag., May, 1000, p. 171.
0.	Finney.	Died.	Philadelphia Med. Journ., March 3, 1000, p. 503.
10.	Garrow.	Recov.	Hamilton, Montreal Med. Journ., Feb., 1001, p. 03.
11.	Harrisson.	"	British Med. Journ., Oct. 20, 1804, p. 805.
12.	Harte.	"	Pennsylvania Hospital Records.
13.	"	"	Ibid.
14.	Le Conte.	Died.	Ibid.
15.	Mitchell, C. F.	Recov.	Ibid.
16.	Mitchell, J. F.	"	Johns Hopkins Hosp. Rep., vol. x., No. 8, p. 453. Case 21.
17.	"	"	Ibid., p. 447. Case 19.
18.	"	Died.	Ibid., p. 450. Case 20.
10.	Mixter.	Recov.	Trans. Amer. Surg. Assoc., 1000, p. 419. Case 23.
20.	McArthur.	Died.	ANNALS OF SURGERY, 1002, vol. xxxvi. (mentioned at p. 022 by Dr. Frank Billings).

No.	Operator.	Result.	Reference.
21.	Munro.	Died.	Boston Med. and Surg. Journ., Feb. 5, 1903. Case 18.
22.	"	"	Ibid. Case 10.
23.	Nimier.	"	Loison, Revue de Chir., Paris, 1901, vol. xxiii. pp. 177 and 188.
24.	Rochard.	Recov.	Bull. et Mém. de la Soc. de Chir. de Paris, dec. 26, 1900, p. 1161.
25.	"	"	Ibid.
26.	Rodman.	Died.	Proceedings of Phila. Acad. of Surgery, April 6, 1903.

REFERENCES.

[1] Morgagni. De sedibus et causis morborum, Epist. i., art. 2. Berthomieu: Annales de la médecine physiologique, Paris, 1822, i. 392–398. Corrin: Archives générales de médecine, Paris, 1831, xxv. 36–60. Ramsay: American Journal of the Medical Sciences, 1836, xviii. 52. Louis: Archives générales de médecine, Paris, 1823, i. 17–49. Howship: Pract. Obs. in Surgery, London, 1816, p. 264, etc.

[2] Practice of Medicine, fourth edition, vol. i. p. 329.

[3] Nothnagel's Encyclopædia, loc. cit.

[4] Principles and Practice of Medicine, New York, 1899.

[5] Pepper's System, loc. cit.

[6] Philadelphia Medical Journal, January 19, 1901.

[7] Yates. American Medicine, May 2, 1903.

[8] Quoted in Thatcher's article on "Typhoid Fever," in the Twentieth Century Practice of Medicine, 1899, vol. xvi. p. 586, New York.

[9] Flexner. American Journal of the Medical Sciences, August, 1903. Yates: American Medicine, May 2, 1903.

[10] T. K. Holmes. Journal of the American Medical Association, March 14, 1903.

[11] Philadelphia Medical Journal, March 3, 1900, p. 505.

[12] Ibid., May 2, 1903.

[13] American Journal of the Medical Sciences, May, 1903.